every complete tear of the perineum should be looked upon as a major problem of the highest order. The possibilities of prevention are very real, and should everywhere be recognized by physicians practicing obstetrics. The suffering caused by these major birth injuries and their remedial difficulties call for meticulous attention to every detail of operative correction.

Department of Obstetrics and Gynecology,
University of Michigan.

USE DESTRUCTION IN THE HUMAN BODY*

By A. W. Meyer, M.D.
Stanford University

With respect to the topic under discussion, I think it is fair to say that we have been in the position of a physiographer, whose eyes are fixed upon earthquakes as the sole factor in topography.

Much has been written about joint mice, for example, the severe pathological process or the violent movement which may produce them, and the sudden painful effects which may accompany them. The fact that an innumerable number of joint mice present in diarthroses cause no symptoms, and that their number can be increased by movement, has been largely forgotten. Neither the production of these nor their presence becomes known to the individual, and except in so far as they were regarded as fragments of hyaline articular cartilage, supposedly contributing to the formation of synovia, no special significance was attached to them. Yet their existence is as significant in regard to the integrity of the articulations concerned as is that of erosion to topography. However, the microscopic and macroscopic particles contained in synovial fluid are not all derived from hyaline cartilage, but come from the capsules, ligaments and fibrocartilage as well, and under unusual conditions may arise from muscles. The presence in synovia, from diarthroses, of particles transitional in size between large incapacitating joint mice and minute microscopic particles, is a matter of special significance and was considered briefly elsewhere.[1] These particles, like the larger incapacitating joint mice, also have a traumatic origin, though not in the customary sense of the word, for they are the products of attrition.

Other bodily changes which I believe to have a similar origin are such phenomena as are discussed in the following:

SUPERFICIAL AND DEEP SYNOVIAL BURSAE

1. Fraying and destruction of the walls of the superficial and deep synovial bursae. These phenomena are commonest in such superficial bursae as those overlying the olecranon and the patella, as shown in Figures 3, 4, 5, 6,[1] and in such deep bursae as those about the shoulder, particularly the subdeltoid or subacromial, and the size of the frayed areas and the depth of the destruction vary greatly. As is well known, there may be a subacromial and a subdeltoid bursa in the same adult shoulder, but there may also be none. Since Black[2] found that the first bursa in this region develops subacromially, that designation would seem preferable. It is such also, because it is absent less rarely than the subdeltoid bursa; but since the latter portion of the single or combined bursae usually is considerably larger, the latter designation represents the usual conditions better.

It would be easy to arrange a progressive series from the well-preserved, smooth, normal bursa to one the entire thickness of the synovial wall of which was completely eroded from within, and the bounding connective tissues, muscles or tendons of which are frayed. These phenomena can be seen even in the bursa of the iliopsoas, as shown in the accompanying Figures 1 and 2, and on the deep surface of the obturator internus, as represented in Figure 3. Slight fraying of the walls of the synovial bursae is invariably present somewhere after the third decade of life, and can easily be recognized without the aid of a hand lens. Indeed, it could be detected in some bursae of a youth of thirteen who had not been unusually active, and in whom related disease could be excluded.

The existence of superimposed bursae, or their formation where only one is normally present, as in the region of the olecranon and in that of the great trochanter, furnishes conditions permitting the partial destruction, fasciculation and fraying of the interbursal walls, as shown in Figures 7[3] and 5.[1] What is surprising, at first, is that all this may happen without the presence of bursal content of any kind, save the usual amount of synovia, and small fragments of the shredded tissues, if the bursae have not enjoyed so long a period of rest that the particles have undergone comminution and lysis.

FRAYING OF ARTICULAR CAPSULES

2. Fraying on the inner surfaces of articular capsules, also referred to in 1915,[4] is the commonest change within articulations. As shown in the case of the capsule of the knee joint represented in Figure 21,[5] this fraying can be very pronounced in an otherwise perfectly normal joint. It is found where the articular capsules come into contact with the margins of the articular cartilages, and the common presence of these capsular fringes somewhere suggests that some of the synovial villi of surgical literature probably belong in this category. This fringing may be present over a considerable area of the inner surfaces of the articular capsules, and usually is more pronounced on the right side of the body, as illustrated in Figures 4 and 5. It often is extremely marked in the radiocarpal articulation, as represented in Figure 20,[5] and is commonly present at the margins of the plica mucosa of the knee. Broader but exceedingly thin capsular tags with frayed margins, the detachment of which would yield free bodies akin to those referred to above, frequently form a part of these fringes.

ARTICULAR CAPSULE DEFECTS

3. In addition to fringes within and without articular capsules, the latter may contain small and large defects. These occur most frequently in the shoulder, the acromioclavicular and the hip joints,

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but they are encountered also at the lateral malleolus, beneath the peroneal tendons and on the plantar surfaces of the first and fifth metatarsophalangeal articulations, as shown in Figures 6 and 7. In these two cases the subcutaneous bursae communicated with the underlying tendon sheaths and the respective articular capsules.

The acromioclavicular capsules also may possess defects below, as reported in 1922,[6] even when the respective humeroscapular capsule is imperforate. Contrary to what I had at first concluded from specimens containing large defects in the superior portion of the humeroscapular articulation, the former are produced more commonly from within by contact with the inferior articular margin of the distal extremity of the clavicle even when the latter is normal in all respects, and they may present a striking appearance, as shown in Figure 8, from above, or as in Figure 16, from below.

Surprisingly large defects may be present in the superior thicker portion of the humeroscapular capsule, as illustrated in Figures 4, 5, and 6,[3] even when the very thin dorsal and inferior portions are intact. They are commonest medial to the greater tuberosity in the region of the supraspinatus tendon, but occur also between the latter and the tendons of the subscapularis and infraspinatus even though the entire width of the tendon of the supraspinatus, even if not its entire thickness, is intact, as represented in Figures 8[6] and 3.[7] In extreme cases, however, the place occupied by this tendon, that of the adjacent portion of the infraspinatus, and that of the tuberosital portion of the subscapularis with the intervening portions of the capsule, may be occupied by a single large defect permitting contact between the humeral head and tuberosities and the deltoid. Defects in the anterior aspect of the capsule of the shoulder joint apparently result from contact with the coracoid, and those in the upper portion from contact with the acromion and clavicle.

DESTRUCTION OF LIGAMENTS

4. The destruction of ligaments. That articular ligaments, even such as the superior and middle glenohumeral and the coracohumeral, must be divided during the formation of large defects in the related portions of the capsules, is self-evident. However, the round ligament of the femur also may be divided or detached at either end, and frayed or be destroyed, except perhaps for some small remnant on the head or in the acetabular fossa, as represented in Figures 9 and 10.

MUSCLE FRAYING

5. There may also be fraying of muscles from movable contact with each other, and from such contact with other structures through defects in articular capsules. Marked fraying of the deep surface of the deltoid after destruction of a considerable portion of the floor of the bursa and the underlying articular capsule, and of the tendon of the supraspinatus, also occurs. It is not surprising that the inner surface of the deltoid may become frayed very seriously, as represented in Figure 11, and that a considerable portion of this muscle may be completely destroyed. Indeed, all that portion of the belly of the muscle overlying the lateral part of the humeral head and the tuberosities may be absent, and represented only by a thin connective tissue membrane, as shown in Figures 12 and 13. Rarely, fraying is met also upon some of the muscles of the forearm, as represented in Figure 1.[7]

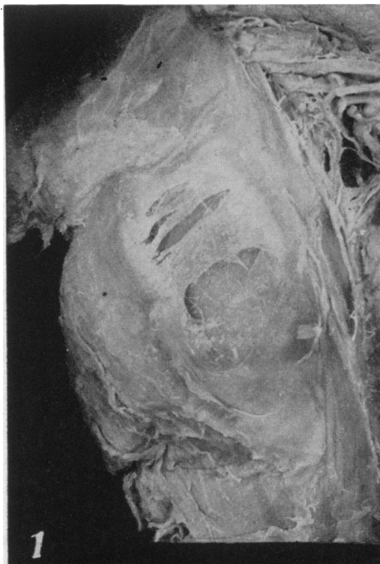
TENDONS

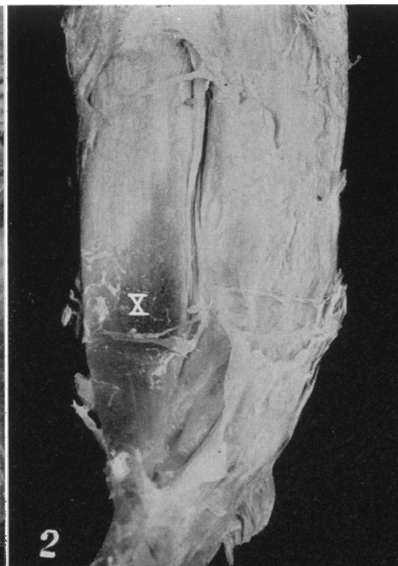
6. Fraying and partial or total division of tendons. Fraying may be present on both surfaces of the tendons of the supraspinatus and subscapularis, on the deep surface of that of the infraspinatus, and the triceps and biceps brachii near their distal attachments. It is met with also on the common flexor and extensor and other tendons of the hand, in those of both the long and short peronei, in the region of the lateral malleolus, and also in the tendon of the long peroneus where, as Edwards[8] showed, it plays upon a facet of the cuboid, except in about 2 per cent of the cases. As represented in Figure 10,[7] even this tendon may be worn deeply and be greatly weakened for a distance of about a centimeter and a half.

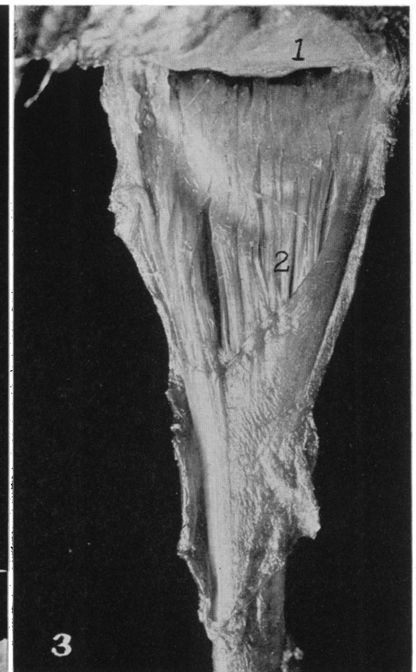
Marked fraying of the deep surface of the tendon of the iliopsoas always was confined to the region in which the articular capsule was defective. Although these defects used to be regarded as developmental, work in this and other laboratories has shown that they are not present in the newborn. Sometimes, when the destruction of the floor of the iliopsoas bursa was marked, there was some indication of fraying on the deep surface of the overlying portion of the tendon, but this fraying always was confined to the synovial and tendon sheaths, and did not affect the tendon fibers. The disproportionate extent of the destruction in the walls of the bursa, shown in Figures 1 and 2, can be ascribed to differences in the relations of the bursal walls. As stated in 1922,[6] the defect in the capsule of the hip joint usually is due to wear from contact with the cartilaginous margin of the femoral head and not with the tendon of the iliopsoas.

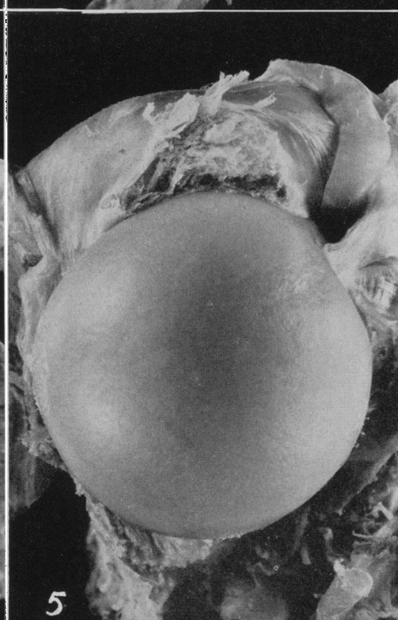
The tendons most commonly and most seriously frayed are those of the supraspinatus and the long tendon of the brachial biceps, both of which frequently are completely divided. There usually is but slight evidence of lengthening of the long tendon of the biceps distal to the humeral tuberosities. This clearly indicates that the sound tendon was not ruptured acutely and retracted by the muscle. This is implied also by the apparent absence of atrophy in that belly, in consequence of lessened use while the tendon was being divided and while it was obtaining a firm secondary attachment. It also is very significant that remnants of the intra-articular portions of the divided tendons are seldom found. This does not imply, however, that a tendon greatly weakened by wear may not yield suddenly under the active pull of the muscle belly or some passive strains to which it may be subjected, or that remnants of the proximal fragment may not survive for some time.

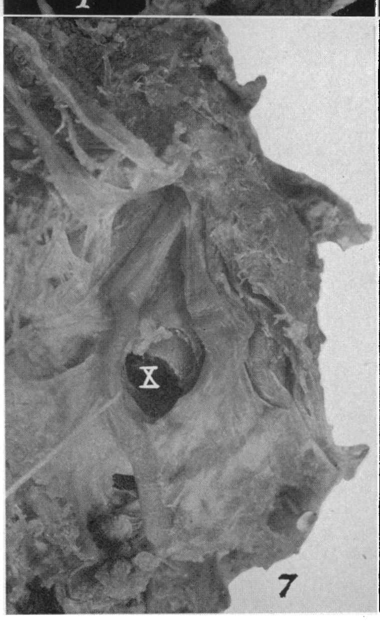
The long tendon of the biceps may be frayed from contact with the capsular sling, represented at *X* in Figure 14, the walls of the intertubercular part of a normal bicipital sulcus, the coracoacromial ligament, the cartilaginous margin of the humeral

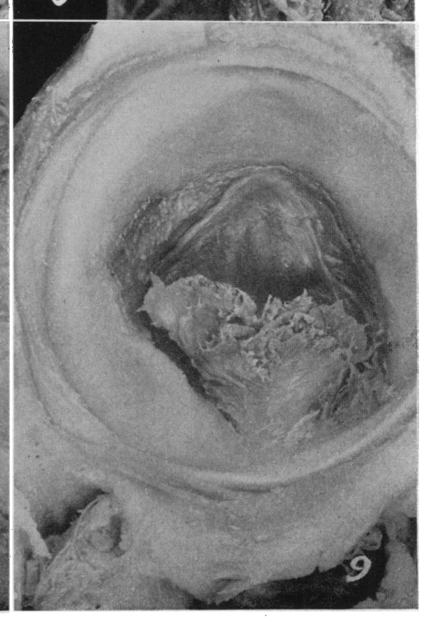












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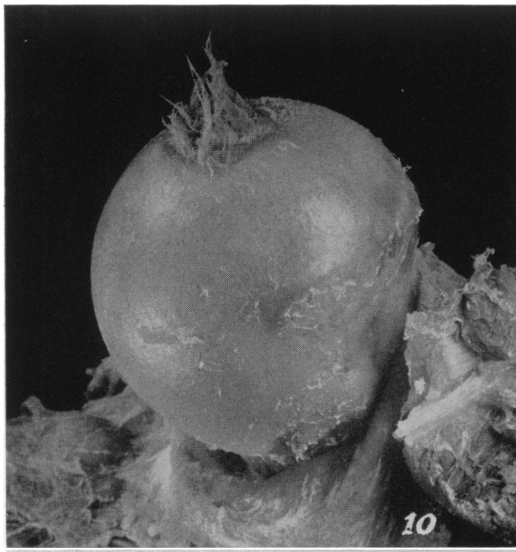

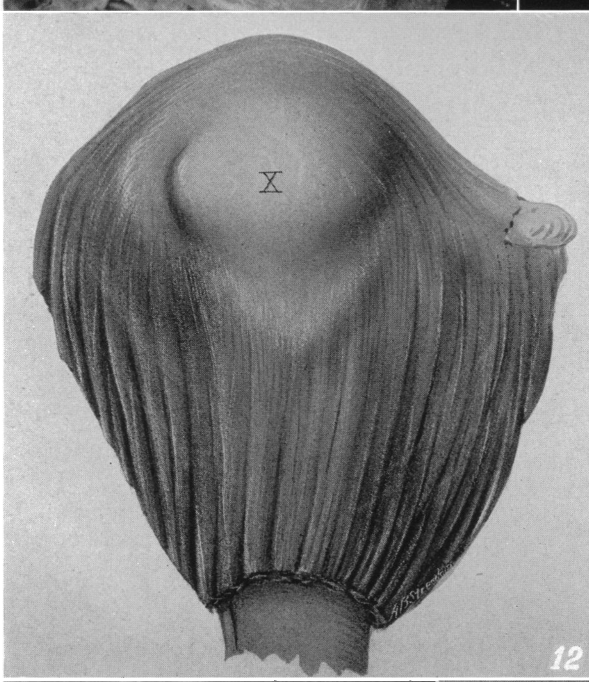

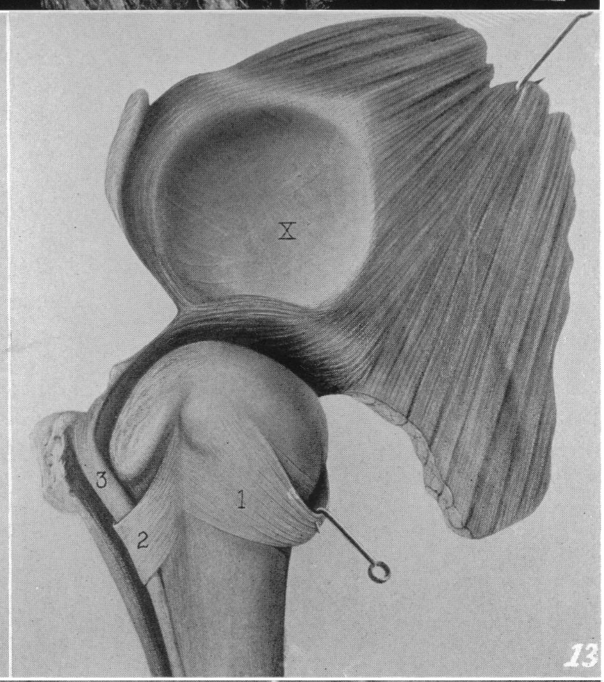

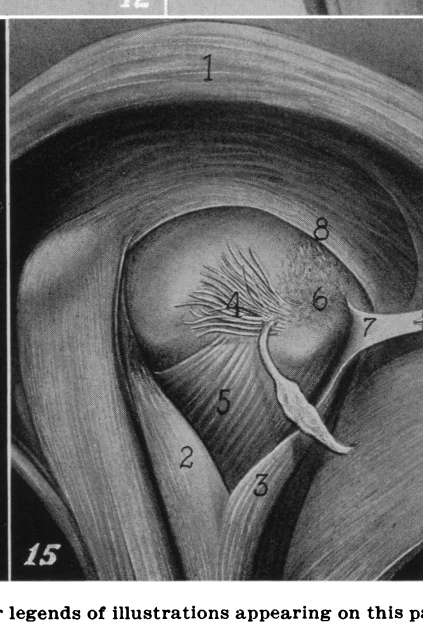

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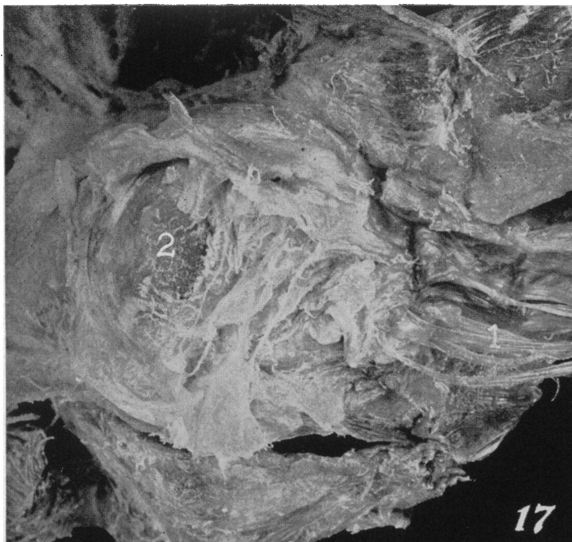

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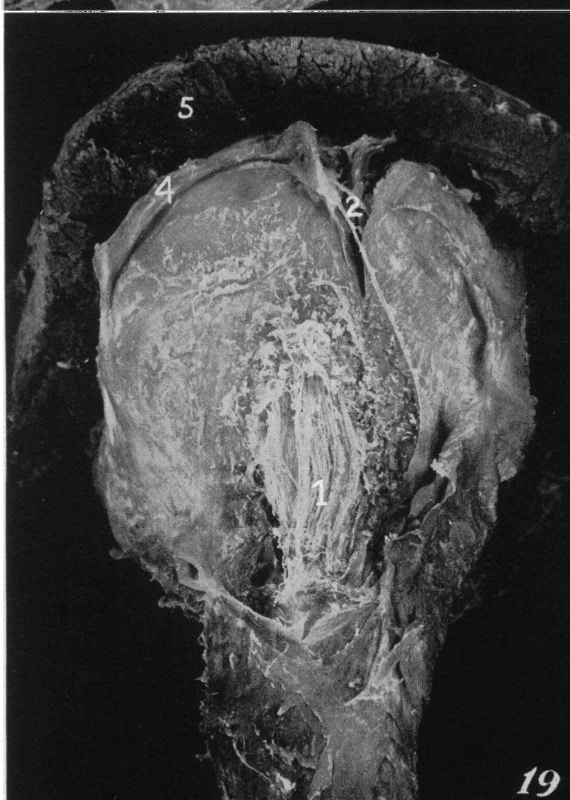

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head, the supratubercular ridge (see Meyer [9]), the capsular roof of the intertubercular sulcus, the lesser tuberosity upon yielding of the capsular sling, and with the tendon of the subscapularis. Its upper surface may also be worn by contact with the acromion and the greater tuberosity, through capsular defects, but I no longer regard this as an important factor in the division of this tendon.

The process of attrition may, to be sure, be hastened by osteophytic reaction of the traumatized periosteum in any region of contact, but it often is effected wholly without it. Division of the tendon seldom results in more than slight elongation of the brachial portion, because the synovial reflection distal to the tuberosities undergoes hypertrophy from the strains to which it is subjected during the gradual slight retraction of the tendon while slowly undergoing dislocation and division. The synovial reflection usually hypertrophies laterally and medially and, as shown in Fig. 15,[3] this may be so marked that it is difficult, if not impossible, to distinguish the acquired attachment from the tendon itself by its outward appearance alone. The fact that the new attachment of the tendon sometimes is hollow for a short distance near its proximal extremity is excellent evidence in support of the assumed process of secondary attachment. This assumption is supported also by the frequent widening of the new attachment, by its location and by the fact that one sometimes finds minor bands extending upward from the divided tendon to the articular capsule, which, no doubt, represent the hypertrophic superficial portion (or the roof) of the synovial sheath of the tendon. The main secondary attachment usually is located just distal to the lesser tuberosity, but it may involve the floor of the sulcus and also the diaphysis near the greater tuberosity.

Among scores of cases of this kind I have never seen a free retracted distal stump of the long tendon. Fraying and partial or complete division of it and secondary attachment may occur without the presence of any defects in the overlying articular capsule or marked fraying of the walls of the subdeltoid bursa, although the tendons of the subscapularis and the supra- and infraspinatus may be frayed and fasciculated. The tendon of the subscapularis sometimes is in this condition on both its superficial and deep surfaces.

The tendon of the supraspinatus, on the other hand, is more frequently and more seriously affected on its superficial surface, and that of the infraspinatus on its deep surface, as represented in Figure 19.[5] The fraying on the superficial surface near the tuberosital attachment of the tendon of the supraspinatus may be from contact with a wholly normal undersurface of the acromial process; but when due to this cause it can include only about an inch of the tendon. The fasciculation and wear on the superficial surface of the more medial portion of this tendon and muscle always were due to contact with the deep surface of the distal extremity of the clavicle through a perforated acromioclavicular articulation, as illustrated in Figures 16 and 19, or with an exostosis on this extremity of the clavicle, acting through an intact

(a) (b)

Fig. 23.—(a) Left half of the cervical and upper thoracic vertebral column with fissuring of the intervertebral discs in the lower cervical region, accompanied by some lipping on the ventral margins of the bodies of some of the vertebrae. (b) A similar view of the lower thoracic and sacral spinal column, showing similar conditions.

acromioclavicular capsule, as was the case of the clavicle shown in Figure 18. The fraying and fasciculation in these two tendons extend for a distance of four inches from the tuberosity in case of the latter, and three inches in case of the former. The differences in the appearances of the lesions are due to the difference between the agents by which they were effected: an uncovered clavicular extremity in the first instance and a capsule-covered exostosis on it in the second.

DISLOCATION OF TENDONS

7. The gradual dislocation of tendons. Instead of being frayed, the long tendon of the biceps may be dislocated ventrally (forward) to a greater or lesser extent, so that it comes to lie in a capsular sling, and if not divided by attrition before that can happen, also upon the tendon of the subscapularis. However, except in extreme cases, it is found *in situ* in the intertubercular sulcus, distal to the tuberosities. The occasional absence of fraying on the deep or superficial surface, or along the ventral (or anterior) and dorsal (or posterior) margins of the tendon may be accounted for by the fact that the underlying and overlying capsule forms a yielding and cushioning sling which moves upon the tendon and protects it against contact with the relatively rough underlying bone. However, as indicated by facts previously mentioned, this does not imply that the attrition between soft parts does not result in fraying.

As emphasized elsewhere,[9] the dislocation of this tendon often is facilitated by the presence of a supratubercular ridge, and whenever the attachment of the underlying portion of the tendinous sling to the humeral head yields in the region ventral (anterior) and proximal to the intertubercular sulcus, the tendon may move forward upon the lesser tuberosity, and finally also upon the tendon of the subscapularis, as represented in Figures 4,[8] 4,[9] and the accompanying Figure 15. In extreme cases of dislocation, the long tendon has not only left the intertubercular portion of the bicipital sulcus, but also the distal portion for a centimeter or two, being prevented from further displacement distally by the absence of oblique traction, by its relation to the tendon of the pectoralis major which often encloses it, and by a strong aponeurotic triangular expansion which may extend upward directly medial to the long tendon fusing with its fascial covering.

Aside from dislocation of the plantar sesamoids and the tendon of the long flexor of the first toe in hallux valgus, the well-known voluntary and involuntary partial, medial dislocation of the tendons of the long and short peronei, and the relatively minor displacement of the tendon of the quadriceps extensor accompanying marked changes in the configuration of the patella and the medial femoral condyles from cartilaginous and osseous destruction, I know of nothing at all comparable to the spontaneous dislocation seen so frequently in case of the long tendon of the biceps.

DESTRUCTION OF FIBROCARTILAGES

8. Destruction of fibrocartilages. Fraying of the inner margins of the glenoid fibrocartilages is practically universal in later years and marked fraying very common then. The genual menisci usually show less fraying of the inner margins, although their deep surfaces may be affected from contact with the hyaline cartilage of the tibial condyles, in spite of the relatively restricted range of movement between them. Besides this fraying, the presence of defects in such articular discs as those of the temperomandibular, the sterno- and acromioclavicular, and the ulnocarpal joints, not only is common, but the acromioclavicular disc usually —not always present—is perforated or largely destroyed. The defect in the ulnar articular disc usually begins in the form of a dorsovolar slit which becomes more rounded as it is enlarged in a radioulnar dirction, and the process of attrition may end in complete destruction of this fibrocartilage.

DESTRUCTION OF HYALINE ARTICULAR CARTILAGES

9. Destruction of hyaline articular cartilages begins upon the surface, not in the depth, and the first thing to suffer is the superficial membranoid layer. Portions of this thin layer several square centimeters in area not infrequently still have a proximal attachment, as shown in Figures 6 and 7.[5] This destruction may continue to the stage of complete denudation, with resultant bony contacts and eburnation. There are, indeed, only a few interosseous diarthroses in which complete destruction of the articular cartilages has not been observed, but the most perplexing lesion is the more or less isolated fissuring such as represented in the intercondylar regions of the femur as shown in Figure 20. Although it is common, especially here and on the patella, I am not certain as to its exact genesis.

10. Fissuring of the intervertebral discs. Clefts, such as represented in Figures 23-a and 23-b and studied especially by Schmorl and his associates,[10] are commonly present in bodies from the later decades, such as used by us for dissection. Since the earliest evidences of fissuring are said to appear in the region of the nucleus pulposus by the seventh year of life, one should not be surprised to find an extension of the process in later decades. The fissuring is mainly transverse, however, and often passes through the middle of the discs. One can usually see some evidence of the process in the third, although the discs may be but slightly affected in the seventh decade. The phenomenon is most frequent and most extensive in places of greatest motion: the lower cervical and the thoracolumbar and lumbosacral regions. It is striking that this correspondence is so good, and surprising that the fissuring often is so complete, and that nothing of the discs may remain except small remnants on the surfaces of the bodies of the coapted vertebra. Such fissuring, attrition and lysis must, to be sure, seriously impair the stability of the spinal column, and must necessarily result in considerable approximation of the bodies of the vertebrae.

Since no two intervertebral discs are identical in structure, and since they are not subjected to identical stresses, one should expect some variation in the location and direction of the fissuring; and this is the case. It is very significant, indeed, that these horizontal fissures not infrequently bisect the discs near the middle of their thickness, where the cleavage stresses are greatest and the nutritive conditions the poorest. Since these fissures, which Luschka regarded as true articulations, very commonly contain fibrocartilaginous fragments, and since the coapted surfaces of the fissured discs are frayed, there can be no doubt that motion is a factor in their production and in the consequent reduction of the discs, and that, to this extent at least, the process in the discs is akin to the other changes here considered. There is much reason to believe that the fissuring is largely, if not wholly, due to a breakdown of the structural materials of which the discs are composed and that it can be adequately explained upon mechanical grounds alone.

EROSION OF BONE

11. Erosion of bone. Whenever coapted areas of the articular cartilages are worn off, enabling the underlying compactae to come into movable contact with each other, they become strengthened by sclerosis of the underlying spongiosa, and are polished and worn, producing the so-called eburnation. Since the compactae on articular surfaces are normally very thin, they would soon be worn through except for the accompanying sclerosis, in response to increasing pressures and friction, while the overlying cartilages are being thinned. It often is forgotten that there also is destruction of bone under these conditions and that the coapted bones may be destroyed to a depth of centimeters in such locations as the knee and hip joints. Even the

radial and ulnar styloids may be destroyed and the underlying portions of the diaphyses of the respective bones deeply worn away and highly polished, through none other than strictly usual movements.

Sclerosis and polishing, commonly called eburnation, and the accompanying erosion, frequently are present also upon the odontoid and the coapted surface of the atlas; between the pisiform and triquetral; the hamate and the lunate; the lunate and the scaphoid and multiangulars; upon the humeral head, in the greater tuberosity, the acromion and glenoid cavity; the patella and the femoral and tibial condyles; the head of the radius and the capitellum, and even upon the dorsal surfaces of the upper ribs from contact with the vertebral borders of the scapulae. This erosion and eburnation may be limited to extremely small areas, but occur wherever bony contacts are possible, provided motion is sufficiently repeated, although the range may be very slight. It is not uncommon to find erosion and eburnation unaccompanied by other bony changes, although slight periosteal reaction may be present at the periphery of the area of erosion of the bones that are being reduced in size through attrition. The process not infrequently is seen also upon heteroplastic bone and on the opposed, overriding, ununited ends of fractured bones. Since the phenomenon is so commonly present, and since the shape of such small bones as the carpals may be completely changed by the reduction of their volume to less than half the normal by attrition, it has always perplexed me that those familiar with these things find it hard to believe that soft parts, such as capsules, ligaments, tendons, and muscles, etc., are affected similarly by the same forces.

RUPTURE OF TENDONS

12. Rupture of tendons. Since structures weakened by partial destruction are not so strong as they were before, it follows that they may rupture suddenly even under relatively moderate strains, but that is quite a different matter than acute rupture of sound tendons.

There was no evidence that any of the fissuring or any of the instances of division of the ligamentum teres of the femur or those of the tendons of the biceps and the supraspinatus, were produced by past dislocations; and since relatively weak fasciculi of these tendons and of the capsules often remain unruptured, apparently for years if not for decades, it is unlikely that unusual, momentary forces were involved in their production. The tendon fasciculi which remained often were so slender that only a weak contraction of the respective muscles could have ruptured them.

FORMATION OF BONY EXCRESCENCES

13. The formation of bony excrescences. Whenever the periosteum is irritated by structures which move upon it, the formation of exostoses can be expected. They frequently are present in a minor form and sometimes in a marked degree, in regions undergoing denudation. Not infrequently, however, there is no indication of any bony change in areas in which great destruction of soft parts or of bone has occurred. The relation of the rate of denudation to the formation of exostoses, in response to mechanical irritation of the periosteum, should need no emphasis.

COMMENT

It is self-evident that such lesions as here considered must have been produced from the time that men and women drew water and hewed wood with sufficient persistence. Hence they must have been seen by the very earliest human anatomists. It would be interesting to consider their production and significance in detail and to examine the historical aspects of the subject, but that is precluded here. I must add, however, that this inventory of the chronic kinetic lesions which often are so pronounced in the later decades of life, is based solely upon material which bore no evidences of pathological processes related to them. I realize that anatomists are not pathologists, but they are not, therefore, ignorant of the gross bodily changes due to accident and disease, and although the number of bodies dissected is relatively small, there are no autopsies comparable in detail to those done in the dissecting room. Here each body can be kept under observation as long as desired, and everything can be examined *ad libitum* both grossly and microscopically. Anyone in doubt regarding the lesions here reported can quickly resolve his doubts by examining the material himself. He need not depend upon what he can learn through small incisions made in the operating or autopsy room.

It should be evident that important articular structures may be weakened or severed by the attrition accompanying ordinary routine movements in the course of daily life. The ultimate effects can, to be sure, be modified and intensified by emergency and extraordinary uses and also by accident and disease, but it is not the greatness of the range but the frequency of the motion that is the important factor. As illustrated in Figures 21 and 22, the effects of attrition are related to handedness and particularly common and pronounced in the bodies of laborers such as carpenters, miners, sailors, charwomen, and housewives. The bodies of those dead from chronic tuberculosis or other long-standing disease usually bore slight evidences of them unless the individual had not been greatly incapacitated by his affliction.

None of the frayed structures bore any evidences of *restitutio ad integrum*. Healing, whenever possible, can occur only when the processes of repair exceed those of destruction. This holds for the acme of life as well as for its decline, and were it not so the affected structures should have been restored to a pristine condition long before death of the affected individuals, which usually occurred after relatively long inactivity and sojourn in an institution.

It always has seemed to me that even very pronounced destruction probably can occur from attrition without severe pain if the rate of erosion be sufficiently slow. Indeed, it is not improbable that the affected individuals mainly become aware of the process when some unusual strain partially detaches or ruptures structures undergoing denudation, or when sudden rupture or fracture finally occurs. This does not imply, to be sure, that infection may not supervene and that its effects could not rapidly manifest themselves.

IN CONCLUSION

Although man must always have been aware of and must also have understood the effect of friction upon his hair, nails, skin, and teeth, and have experienced intertrigo, this is not true regarding the internal evidences of attrition. The latter not only have been largely overlooked by our profession, but seem to tax the credulity of some of us even today. This is due to the fact that anatomists alone have full opportunity to observe them. The rank and file of physicians who merely have done a dissection of one side of a body of one kind or another have entirely too narrow a basis for judgment. Pathologists feel fortunate if they may open the abdomen, thorax and skull, or occasionally a joint, and most orthopedic surgeons only have such additional knowledge as can be obtained through small incisions upon the living or a fleeting visit to a dissecting room. Moreover, it is an old assumption that human joints are frictionless and that hyaline cartilages do not wear. It often is assumed that slight injuries done to structures coapted intimately during movements are repaired quickly during the customary periodic rests. Although this might be the case in such highly vascular structures as synovial bursae and articular capsules in the young, it can hardly apply to such structures as the cartilages, both hyaline and fibro-, for they are practically avascular. And although healing may occur in an avascular structure such as the cornea, regeneration does not take place even in it, although bathed constantly by a fluid medium. In any case, whether repair exceeds wear depends upon the relative rates of the two processes and only secondarily upon the age, the vascularity, the structure and the nutrition of the organ in question, and healing cannot occur in avascular, hyalinized, separated tags of capsules, ligaments, and tendons.

Department of Anatomy, Stanford University.

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LEGENDS

Fig. 1.—The ventral surface of the capsule of the right hip joint, containing defects with frayed margins, in the deep wall of the psoas bursa.

Fig. 2.—The deep surface of the respective iliopsoas tendon, showing slight fraying of the coapted synovial surface at (X).

Fig. 3.—The inferior surface (1) of a lesser ischiatic incision, and the coapted surface (2) of the right obturator internus muscle, showing considerable local destruction of the synovial and tendon sheaths, and some fasciculation of the tendon.

Fig. 4.—A right supraspinatus tendon (above) reflected laterally and viewed from below, to show the marked fraying of the synovial membrane, the articular capsule and the undersurface of the tendon from contact with the wholly normal and smooth cartilaginous margin of the head of the humerus.

Fig. 5.—A similar view of the paired tendon from the same body, showing a smaller area and a lesser degree of wear (above). The proximally divided long tendon of the biceps is also reflected.

Fig. 6.—A defect (X) in the plantar portion of the capsule of the first metatarsophalangeal articulation in a left foot, with moderate hallux valgus.

Fig. 7.—A similar defect (X) in the same portion of the articular capsule of the left fifth metatarsophalangeal articulation. The long flexor tendon was drawn medially.

Fig. 8.—A defect (X) in the inferior portion of a right acromioclavicular capsule, viewed from above, after removal of the clavicle, the inferior margin of the distal extremity of which was normal.

Fig. 9.—A right acetabulum, with a proximally detached and frayed round ligament.

Fig. 10.—The head of the accompanying femur with a remnant of the round ligament still attached and slight fraying of the articular cartilage.

Fig. 11.—The deep surface of a right deltoid with frayed sheath and muscle fasciculi, from contact with the humeral head and tuberosities, from a case with destruction of the upper portion of the articular capsule and the included tendon of the supraspinatus.

Fig. 12.—A right shoulder represented from the side, with the deltoid exposed, revealing a large central area (X) composed only of a thin, nonmuscular membrane.

Fig. 13.—The same shoulder drawn with the deltoid reflected, to show the membranous area (X) from within, also revealing complete destruction of the upper portion of the articular capsule with its contained tendons, and maximal dislocation forward of the tendon of the long head. The upper margins of what remains of the capsule are indicated at (1) and (2), and the tendon of the long head at (3).

Fig. 14.—A picture of a lateral view of a right humerus, upon which the long tendon of the biceps had been dislocated forward considerably, showing the opened capsular sling, the deep portion (X) of which had retained its proximal attachment to the head and neck, but had lost it over the lesser tuberosity.

Fig. 15.—A lateral view (drawing) of a left humeroscapular articulation with the deltoid (1) cut away to reveal a large defect in the upper portion of the articular capsule, a markedly dislocated long tendon (2), its greatly hypertrophied lateral synovial reflection (3), the remnants of the upper portion of the tendon of the subscapularis (4), the fasciculated residual attached portion of this tendon (5), the roughened upper surface of the lesser tuberosity (6), the slightly retracted portion of the synovial sheath, beneath which the long tendon originally lay (7), and the thinned, free, crescentic lateral border (8) of the capsule and the tendon of the supraspinatus.

Fig. 16.—An acromioclavicular articulation seen from below, revealing a defect (X) in the inferior portion of the capsule and (3) part of the inferior margin of the distal extremity of the clavicle.

Fig. 17.—A supraspinatus tendon seen from above, showing coarse fasciculation of the muscle and tendon (1) and the tuberosital region (2) of the same tendon, with considerable superficial fraying. The latter was produced by contact with the undersurface of the acromion, and the former by the capsule-covered exostosis (X, Figure 18) which pressed against the tendon and muscle of the supraspinatus.

Fig. 18.—The distal extremity of the said right clavicle, seen from below, showing the exostosis (X), which was intracapsular.

Fig. 19.—The upper surface of the supraspinatus tendon and muscle (belonging with clavicle shown in Figure 16), with a markedly frayed, finely fascicular portion at (1), from contact with the practically normal distal extremity of the clavicle shown at 3 in Figure 16. There, also, is some fraying of the distal portion of this supraspinatus tendon from contact with the inferior surface of the acromion. The opened bicipital sulcus is seen from above at (2), the reflected roof of the subdeltoid bursa at (4), and the cut surface of the deltoid at (5).

Fig. 20.—Fissuring of the hyaline articular cartilage on the distal extremity of a right femur with slight fraying of the former.

Fig. 21.—A pair of biceps tendons illustrating the greater wear in the right (upper) tendon in right-handed persons.

Fig. 22.—Another pair of similar tendons emphasizing the same thing with the right tendon below.